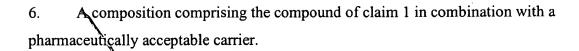
What is claimed is:

1. A compound of formula (I):

wherein R^1 is lower alkyl, lower alkenyl, (hydroxy)lower alkyl, lower alkynyl, phenyl, benzyl or 2-thienyl, R^2 , R^3 , R^4 and R^5 are the same or different and are each hydrogen or lower alkyl; each R^6 is individually hydrogen, lower alkyl, hydroxy, (hydroxy)lower alkyl, lower alkoxy, benzyloxy, lower alkanoyloxy, nitro or halo, n is 1-3, R^7 is hydrogen, lower alkyl or lower alkenyl, X is oxy and thio, Y is carbonyl, $(CH_2)_{1-3}$, $(CH_2)_{1-3}SO_2$ or $(CH_2)_{1-3}C(O)$, and Z is $(\omega$ -(4-pyridyl)(C_2 - C_4 alkoxy), $(\omega$ -($(R^8)(R^9)$ amino)(C_2 - C_4 alkoxy), wherein R^8 and R^9 are each H, $(C_1$ - C_3)alkyl or together with N are a 5- or 6-membered heterocyclic ring comprising 1-3 $N(R^8)$, S or nonperoxide O; an amino acid ester of $(\omega$ -(HO)(C_2 - C_4))alkoxy, $N(R^8)CH(R^8)CO_2H$, 1'-D-glucuronyloxy; or Y-Z is $(CH_2)_{1-3}R^8$; wherein R^8 is OH, $(C_2$ - C_4)acyloxy, SO_3H , PO_4H_2 , N(NO)(OH), SO_2NH_2 , $RO(OH)(NH_2)$, or tetrazolyl; or a pharmaceutically acceptable salt thereof.

- 2. The compound of claim 1 wherein Z is the L-valine or L-glycine ester of 2-hydroxyethoxy.
- 3. The compound of claim I wherein Z is N-morpholinoethoxy.
- 4. The compound of claim 1 wherein each R⁸ is H, CH₃ or i-Pr.
- 5. The compound of claim 1 wherein Z is OCH₂CH₂N(CH₃)₃⁺.

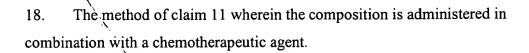


- 7. The composition of claim 6 which is a tablet, granule or capsule.
- 8. The composition of claim 6 wherein the carrier is an aqueous vehicle.
- 9. The composition of claim 8 which is an aqueous solution.

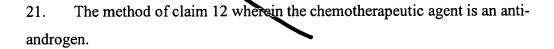


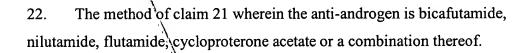
10. A method of inhibiting cancer comprising administering an effective amount of the compound of claim 1 to a mammal afflicted with cancer.

- 11. A method of inhibiting cancer comprising administering an effective amount of the composition of claim 6 to a mammal afflicted with cancer.
- 12. The method of claim 10 or 11 wherein the cancer is prostate cancer.
- 13. The method of claim 10 or 11 wherein the cancer is multiple myeloma.
- 14. The method of claim 10 or 11 wherein the cancer is chronic lymphocytic leukemia.
- 15. The method of claim 11 wherein the composition is administered orally.
- 16. The method of claim 15 wherein an enterically coated dosage form is administered.
- 17. The method of claim 11 wherein the composition is administered parenterally.



- 19. The method of claim 12 wherein the composition is administered in combination with a chemotherapeutic agent.
- 20. The method of claim 18 wherein the chemotherapeutic agent is mitoxanthrone, prednisone, estramustine, vinblastine or a combination thereof.

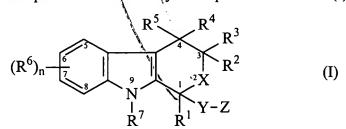




23. The method of claim 21 wherein the anti-androgen is leuprolide acetate, goserelin acetate or a combination thereof.

24. A therapeutic method comprising:

(a) evaluating the level of at least one of PPAR-γ, Mcl-1 or Bag-1 in cancer cells isolated from a patient afflicted with prostate cancer to determine if said level is sufficiently high so that said cells would be susceptible to inhibition by a compound of formula (I):



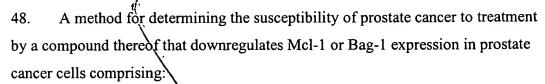
wherein R¹ is lower alkyl, lower alkenyl, (hydroxy)lower alkyl, lower alkynyl, phenyl, benzyl or 2-thienyl, R², R³, R⁴ and R⁵ are the same or different and are each hydrogen or lower alkyl; each R⁶ is individually hydrogen, lower alkyl, hydroxy, (hydroxy)lower alkyl, lower alkoxy, benzyloxy, lower alkanoyloxy, nitro or halo, n is 1-3, R³ is hydrogen, lower alkyl or lower alkenyl, X is oxy and thio, Y is carbonyl, (CH₂)1-3, (CH₂)1-3SO₂ or (CH₂)1-3C(O), and Z is (ω-(4-pyridyl)(C₂-C₄ alkoxy), (ω-((R³)(R³) amino)(C₂-C₄ alkoxy), wherein R³ and R³ are each H, (C₁-C₃)alkyl or together with N are a 5- or 6-membered heterocyclic ring comprising 1-3 N(R³), S or nonperoxide O; an amino acid ester of (ω-(HO)(C₂-C₄))alkoxy, N(R³)CH(R³)CO₂H, 1′-D-glucuronyloxy; or Y-Z is (CH₂)1-3R³ wherein R³ is OH, (C₂-C₄)acyloxy, SO₃H, PO₄H₂, N(NO)(OH), SO₂NH₂, P(O)(OH)(NH₂) or tetrazolyl; or a pharmaceutically acceptable salt thereof; and

- (b) administering to said patient an amount of a compound of formula (I) effective to inhibit said cells or to sensitize said cells to inhibition by administration of a chemotherapeutic agent.
- 25. The method of claim 24 wherein Y-Z is a pyridylalkyl ester, a morpholinoalkyl ester, an aminoalkyl ester or a hydroxyalkyl ester.
- 26. The method of claim 24 wherein Y₄Z is a glucamine ester or N-(C₁-C₄)alkyl-glucamine ester of CH₂CO₂H.
- 27. The method of claim 24 wherein Y-Z is the 1'-D-glucuronate ester of CH₂CO₂H.
- 28. The method of claim 24 wherein Y-Z is a water-soluble amide of CH₂CO₂H.

- 29. The method of claim 28 wherein Y-Z is an amino acid amide of CH₂CO₂H.
- 30. The method of claim 24 wherein the level of PPAR- γ is evaluated.
- 31. A method for determining the ability of a test agent to inhibit prostate cancer cells comprising contacting a population of cells from a prostate cancer cell line that expresses PPAR- γ with said agent and determining whether the agent increases the expression of PPAR- γ in said cells.
- 32. A method for determining the ability of a test agent to inhibit prostate cancer cells comprising contacting a population of cells from a prostate cancer cell line that expresses Mcl-1 or Bag-1 with said agent and determining whether the agent decreases the expression of Mcl-1 in said cells.
- 33. A method for determining the ability of a test agent to inhibit cancer comprising determining whether or not the agent competitively inhibits the receptor-mediated binding of radiolabeled etodolac to cancer cells.
- 34. The method of claim 33 wherein the cancer cells are etodolac sensitive.
- 35. The method of claim 33 wherein the etodolac is R(-)-etodolac.
- 36. The method of claim 31, 32, 33, 34 or 35 further comprising determining whether or not the agent increases the uptake of calcium by cancer cells.
- 37. The method of claim 36 further comprising determining whether or not the test agent can induce a chemokinetic response in a population of lymphocytes.
- 38. The method of claim 37 wherein the response enhances the ability of the lymphocytes to exhibit chemotaxis.

- 39. The method of claim 38 wherein the lymphocytes comprise B-CLL lymphocytes.
- 40. The method of claim 37 further comprising determining whether or not the test agent can induce apoptosis in cancer cells.
- 41. The method of claim 40 wherein the cancer cells are CLL cells.
- 42. The method of claim 40 comprising determining whether or not the test agent can increase caspase-3 activity.
- 43. The method of claim 40 further comprising determining whether or not the test agent lowers the white cell count of a test animal.
- 44. The method of claim 43 wherein the test animal is a mouse.
- 45. The method of claim 43 further comprising determining whether or not the test agent can inhibit cancer induction in the pristane-induced murine MLL model.
- 46. The method of claim 43 further comprising determining whether or not the test compound can inhibit cancer in the transgenic adenocarcinoma mouse prostate cancer model.
- 47. A method for determining the susceptibility of prostate cancer to treatment by a compound that activates PPAR-γ expression in prostate cancer cells comprising:
 - (a) isolating prostate cancer cells from a human subject; and
 - (b) evaluating whether or not said cells express PPAR-γ at a level sufficient to render them subject to inhibition by said compound.

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- (a) isolating prostate cancer cells from a human subject;
- (b) evaluating whether or not said cells express Mcl-1 at a level sufficient to render them subject to inhibition by said compound.

add add E3